10551348
File 5:Biosis Previews(R) 1926-2008/Sep W3
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Set Items Description
--- ----- -----------
? s thymosin and (adhesive or fibrin? or collagen)
2466 THYMOSIN
21900 ADHESIVE
82423 FIBRIN?
123892 COLLAGEN
S1 34 THYMOSIN AND (ADHESIVE OR FIBRIN? OR COLLAGEN)
? s s1 and (fusion or chimer?)
34 S1
115548 FUSION
45196 CHIMER?
S2 0 S1 AND (FUSION OR CHIMER?)
? t s4/7/1-34

S4/7/1-34
>>>Set 4 does not exist
? t s1/7/1-34

1/7/1
DIALOG(R)File 5:Biosis Previews(R)
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0020463681 BIOSIS NO.: 200800510620
Up-regulation of %%%thymosin%%% beta A is a determinant of the transformed phenotype and invasiveness of mouse fibrosarcoma cells
AUTHOR: Nummela P (Reprint); Yin M; Kielosto M; Leaner V; Birrer M J; Holtta E
AUTHOR ADDRESS: Haartman Inst, Helsinki, Finland**Finland
JOURNAL: EJC Supplements 6 (9): p77-78 JUL 2008 2008
CONFERENCE/MEETING: 20th Meeting of the European-Association-for-Cancer-Research Lyon, FRANCE July 05 -08, 2008; 20080705
SPONSOR: European Assoc Canc Res
ISSN: 1359-6349
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

1/7/2
DIALOG(R)File 5:Biosis Previews(R)

0020022416 BIOSIS NO.: 200800069355
%%%Thymosin%%% beta 4 is not always the main beta-%%%thymosin%%% in mammalian platelets
BOOK TITLE: Annals of the New York Academy of Sciences
AUTHOR: Huff Thomas; Mueller Christian S G; Hannappel Ewald (Reprint)
BOOK AUTHOR/EDITOR: Goldstein AL (Editor); Garaci E (Editor)
AUTHOR ADDRESS: Univ Erlangen Nurnberg, Inst Biochem, Fahrstr 17, D-91054 Erlangen, Germany**Germany
AUTHOR E-MAIL ADDRESS: eh@biochem.uni-criangen.de
SERIES TITLE: ANNALS OF THE NEW YORK ACADEMY OF SCIENCES 1112 p451-457 2007
ITEM IDENTIFIER: doi:10.1196/annals.1415.029
BOOK PUBLISHER: BLACKWELL PUBLISHING, 9600 GARSINGTON RD, OXFORD OX4 2DQ, OXEN, UK
CONFERENCE/MEETING: 1st International Symposium on Thymosins in Health and Disease Washington, DC, USA 2007,
ISSN: 0077-8923_(print) ISBN: 978-1-57331-701-6 (S)
DOCUMENT TYPE: Book Chapter; Meeting
RECORD TYPE: Abstract
LANGUAGE: English
ABSTRACT: beta-thymosins constitute a family of highly conserved 5-kDa polypeptides. %%%Thymosin%%% beta(4), the most abundant member of this family, is expressed in most mammalian cell types and is regarded as the main intracellular G-actin sequestering peptide. In addition to this important intracellular function several other activities have been attributed to this peptide. %%%Thymosin%%% beta(4) is released from human platelets and cross-linked to %%%fibrin%%% after activation of platelets with thrombin. While in most mammalian tissues %%%thymosin%%% beta(4) is accompanied by a second member of this peptide family, in human platelets only %%%thymosin%%% beta(4) is present. To elucidate if it is common to mammalian platelets that only one beta-%%%thymosin%%% is present, we analyzed platelets from several mammals for their beta-%%%thymosin%%% content. In human platelets only %%%thymosin%%% beta(4) could be detected, whereas in bovine platelets %%%thymosin%%% beta(9), which is normally the minor P-%%%thymosin%%% in bovine tissues, was identified as the main beta-%%%thymosin%%%. In rabbit platelets, %%%thymosin%%% beta(4) is not simply replaced by the Ala most homologous %%%thymosin%%% beta(4), as might be expected from sequence homology. %%%Thymosin%%% beta(Ala)(4) and %%%thymosin%%% beta(10) were found, but %%%thymosin%%% beta(10) is present in about 2.5-fold higher amounts. Because %%%thymosin%%% beta(Ala)(4) possesses about threefold higher affinity to G-actin, compared to %%%thymosin%%% beta(4), beta(10), and beta(9), we suggest that expression of beta-thymosins is triggered by functional requirements and not sequence homology.

1/7/3
DIALOG(R)File 5:Biosis Previews(R)

0020022411 BIOSIS NO.: 200800069350
Pressure ulcers - A role for %%%thymosin%%% beta 4
BOOK TITLE: Annals of the New York Academy of Sciences
AUTHOR: Godschalk Michael Francis (Reprint)
BOOK AUTHOR/EDITOR: Goldstein AL (Editor); Garaci E (Editor)
AUTHOR ADDRESS: Virginia Commonwealth Univ, Sch Med, McGuire Vet Affairs Med Ctr, 151,1201 Broad Rock Blvd, Richmond, VA 23249 USA**USA
AUTHOR E-MAIL ADDRESS: Michael.Godschalk@VA.gov
SERIES TITLE: ANNALS OF THE NEW YORK ACADEMY OF SCIENCES 1112 p413-417 2007
ITEM IDENTIFIER: doi:10.1196/annals.1415.049
BOOK PUBLISHER: BLACKWELL PUBLISHING, 9600 GARSINGTON RD, OXFORD OX4 2DQ, OXEN, UK
CONFERENCE/MEETING: 1st International Symposium on Thymosins in Health and Disease Washington, DC, USA 2007,
ISSN: 0077-8923_(print) ISBN: 978-1-57331-701-6 (S)
DOCUMENT TYPE: Book Chapter; Meeting
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Pressure ulcers occur in up to 14% of acute hospitalizations. They cause pain, decreased quality of life, increased morbidity, and prolonged hospitalizations. Treatment includes pain control, nutritional support, relieving pressure, removing devitalized tissue, and by using dressings and medications, providing an environment in which healing can occur. Even with optimal treatment, pressure ulcers may take months to heal. %%%Thymosin%%% beta 4 is being investigated as a treatment for pressure ulcers. %%%Thymosin%%% beta 4 has wound healing and anti-inflammatory properties. It is thought to exert its therapeutic effect through promotion of keratinocyte and endothelial cell migration, increased %%%collagen%%% deposition, and stimulation of angiogenesis. A study in a rat full-thickness wound model showed that treatment with %%%thymosin%%% beta 4 increased %%%collagen%%% deposition and angiogenesis and stimulated keratinocyte migration and reepithelialization. If %%%thymosin%%% beta 4 decreases healing time, this would represent a significant advance in the treatment of pressure ulcers.

1/7/4

0020022386 BIOSIS NO.: 200800069325
%%%Thymosin%%% beta(4) upregulates the expression of hepatocyte growth factor and downregulates the expression of PDGF-beta receptor in human hepatic stellate cells
BOOK TITLE: Annals of the New York Academy of Sciences
AUTHOR: Barnaeva Elena; Nadezhda Agladze; Hannappel Ewald; Sjogren Maria H; Rojkind Marcos (Reprint)
BOOK AUTHOR/EDITOR: Goldstein AL (Editor); Garaci E (Editor)
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SERIES TITLE: ANNALS OF THE NEW YORK ACADEMY OF SCIENCES 1112 p154-160 2007
ITEM IDENTIFIER: doi:10.1196/annals.1415.035
BOOK PUBLISHER: BLACKWELL PUBLISHING, 9600 GARSINGTON RD, OXFORD OX4 2DQ, OXEN, UK
CONFERENCE/MEETING: 1st International Symposium on Thymosins in Health and Disease Washington, DC, USA 2007,
ISSN: 0077-8923_(print) ISBN: 978-1-57331-701-6 (S)
DOCUMENT TYPE: Book Chapter; Meeting
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Hepatic stellate cells (HSCs) are the main producers of type I %%%collagen%%% in the liver, and therefore are responsible, in part, for the fibrous sear observed in cirrhotic livers. Although there is no approved treatment for this deadly disease, drugs inducing HSC apoptosis in animals (gliotoxin) and hepatocyte regeneration in man (hepatocyte growth factor [HGF], have been used successfully in ameliorating liver fibrosis. In this communication we investigated whether %%%thymosin%%% beta(4) (T beta(4)), an actin-sequestering peptide that prevents scarring of the heart after a myocardial infarction and that prevents kidney fibrosis in animals, has the potential to be used to treat liver fibrosis. To this end we studied whether the administration of T beta(4) to HSCs could alter the expression of genes encoding for extracellular matrix components, as well as those required for differentiation of HSCs. Our preliminary findings show that T beta(4) had no effect on the expression of alpha 2 (I) %%%collagen%%%, tissue inhibitor of metalloproteinases-1, and matrix metalloproteinase-2 mRNAs. However, it upregulated the expression of HGF and downregulated the expression of platelet-derived growth factor-beta receptor mRNAs in these cells. Overall, these findings suggest that T beta(4) has antifibrogenic potential.

1/7/5
DIALOG(R)File 5:Biosis Previews(R)

0020022384 BIOSIS NO.: 200800069323
%%%Adhesive%%% and proteolytic phenotype of migrating endothelial cells induced by %%%thymosin%%% beta-4
BOOK TITLE: Annals of the New York Academy of Sciences
AUTHOR: Cierniewski Czeslaw S (Reprint); Malinowski Mariusz; Bednarek Radoslaw; Cierniewska-Cieslak Aleksandra
BOOK AUTHOR/EDITOR: Goldstein AL (Editor); Garaci E (Editor)
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SERIES TITLE: ANNALS OF THE NEW YORK ACADEMY OF SCIENCES 1112 p123-139 2007
ITEM IDENTIFIER: doi:10.1196/annals.1415.019
BOOK PUBLISHER: BLACKWELL PUBLISHING, 9600 GARSINGTON RD, OXFORD OX4 2DQ, OXEN, UK
CONFERENCE/MEETING: 1st International Symposium on Thymosins in Health and Disease Washington, DC, USA 2007,
ISSN: 0077-8923_(print) ISBN: 978-1-57331-701-6 (S)
DOCUMENT TYPE: Book Chapter; Meeting
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The early stages of angiogenesis are usually accompanied by the occurrence of vascular leakage, and the deposition of %%%fibrin%%% in extravascular spaces. Initially, the %%%fibrin%%% network acts as a sealing matrix, but later on also as a scaffolding for invading endothelial cells. This process is induced by angiogenic growth factors, particularly by vascular endothelial growth factor (VEGF). Angiogenesis involves proteolytic activities, in particular cell-bound urokinase/plasmin and matrix metalloproteinase (MMPs) activities that modulate the %%%fibrin%%% structure and affect adhesion and migration of endothelial cells. Recent data show that formation of new vessels may be stimulated by %%%thymosin%%% beta-4 (T beta-4), but it is still not clear whether T beta-4 alone is angiogenic or the angiogenic potential of T beta-4 is mediated by VEGF. In this report to further characterize T beta-4 angiogenic activity, we produced its mutants that were deprived of the N-terminal tetrapeptide AcSDKP (T beta-4((AcSDKPT/4A))), the actin-binding sequence KLKKTET (T beta-4((KLKKTET/7A))) and with the nuclear localization sequence damaged by a point mutation Lys16A1a (T beta-4((K16A))). Then we tested their activity to induce expression and

release of MMPs as well as plasminogen activators inhibitor type-1 (PAI-1). We also analyzed their effect on migration and proliferation of endothelial cells in three-dimensional (313) %%%fibrin%%% matrix as well as on their ability to stimulate the outgrowth of human endothelial cells in capillary-like tubular structures. Our data demonstrate that increased intracellular expression of T beta-4 and its mutants is necessary and sufficient to induce PAI-1 gene expression in endothelial cells. Similarly, they stimulate expression and release of MMP-1, -2, and -3. As evaluated by using specific inhibitors to these MMPs, they modified specifically the structure of %%%fibrin%%% and thus facilitated migration of endothelial cells. To sum up, our data show that the mechanism by which T beta-4 induced transition of endothelial cells from quiescent to proangiogenic phenotype is characterized by increased expression of PAI-1 and MMPs did not require the presence of the N-terminal sequence AcSDKP, and depended only partially on its ability to bind G-actin or to enter the nucleus.

1/7/6

0020001978 BIOSIS NO.: 200800048917
Identification of genes specifically expressed by human Muller cells by use of subtractive hybridization
AUTHOR: Lupien Caroline B; Salesse Christian (Reprint)
AUTHOR ADDRESS: CHUQ, Ctr Rech, Unite Rech Ophtalmol, Salle S-5,Pavillon CHUL,2705 Boul Laurier, Ste Foy, PQ G1V 4G2, Canada**Canada
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JOURNAL: Molecular Vision 13 (204-08): p1828-1841 OCT 2 2007 2007
ISSN: 1090-0535
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Purpose: MUller cells are the predominant type of glial cells in the retina. They play a critical role in the retina. The purpose of this study was to generate a profile of the genes specifically expressed by human retinal Muller cells and to identify genes that may be responsible for retinal diseases.Methods: Subtractive hybridization is a method process by which two populations of mRNA are compared in order to obtain clones of genes expressed in one population but not in the other. A cDNA subtraction library was constructed using RNA isolated from human MUller cells and human astrocytes. PCR-select differential screening was used to further verify the differentially expressed cDNA clones. Positive clones were sequenced and analyzed using the NCBI BLASTN program to identify

sequence homologies.Results: We identified 194 clones specifically expressed in human Muller cells. Among these clones, 102 corresponded to known human genes. Of the remaining 94 clones, 75 corresponded to expressed sequence tags or genomic clones and 19 transcripts did not match with any sequence in databases, and are possibly novel genes.Conclusions: The analysis of the subtraction library revealed genes that are specifically expressed by human Muller cells. Some of these genes are unidentified, novel genes that are specific to MUller cells as determined by RT-PCR and Northern blot analyses. These novel genes thus represent candidate genes for retinal diseases.

1/7/7
DIALOG(R)File 5:Biosis Previews(R)

0019812343 BIOSIS NO.: 200700472084
Longitudinal analysis of gene expression in porcine skeletal muscle after post-injection local injury
AUTHOR: Ferre Pierre J; Liaubet Laurence; Concordet Didier; SanCristobal Magali; Uro-Coste Emmanuelle; Tosser-Klopp Gwenola; Bonnet Agnes; Toutain Pierre-Louis; Hatey Francois; Lefebvre Herve P (Reprint)
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JOURNAL: Pharmaceutical Research (Dordrecht) 24 (8): p1480-1489 AUG 2007 2007
ITEM IDENTIFIER: doi:10.1007/s11095-007-9266-8
ISSN: 0724-8741
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Purpose. The purpose of this study is to describe the time course of gene expression in a skeletal muscle local injury induced by an intramuscular (IM) injection, and to compare the dynamics of gene expression with pathological events.Materials and Methods.Ten piglets received 4 IM injections of propylene glycol in the longissimus dorsi muscles 6 h, 2, 7, and 21 days before euthanasia, where control and injected muscle sites were sampled for RNA isolation and microscopic examination. The hybridization of nylon cDNA microarrays was carried out with radioactive probes obtained from the muscle RNA.Results. 153 genes were found under- or over-expressed at least once among the investigated time-conditions. The eight most discriminant genes were also identified: Two genes (GTP-binding protein RAD and Ankyrin repeat domain protein) were over-expressed at 6 h and six genes between 2 and 21 days

(Osteonectin, Fibronectin, Matrix metalloproteinase-2, %%%Collagen%%% alpha 1(I) chain, %%%Collagen%%% alpha 2(I) chain, and %%%Thymosin%%% beta-4). Necrosis, inflammation and regeneration were observed through both the dynamics of gene expression profiles and through the microscopic examinations.Conclusion. Our data demonstrate that several pathways are involved in post-injection muscle injury, and that necrosis, inflammation and regeneration are not sequential but occur in parallel.

1/7/8
DIALOG(R)File 5:Biosis Previews(R)

0019807511 BIOSIS NO.: 200700467252
Current prostate cancer: 20 years later
AUTHOR: Marberger Michael (Reprint)
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JOURNAL: BJU International 100 (Suppl. 2): p11-14 JUL 2007 2007
ISSN: 1464-4096
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Citation
LANGUAGE: English

1/7/9
DIALOG(R)File 5:Biosis Previews(R)

0019801641 BIOSIS NO.: 200700461382
Decreased endogenous levels of Ac-SDKP promote organ fibrosis
AUTHOR: Cavasin Maria A (Reprint); Liao Tang-Dong; Yang Xiao-Ping; Yang James J; Carretero Oscar A
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JOURNAL: Hypertension (Baltimore) 50 (1): p130-136 JUL 2007 2007
ITEM IDENTIFIER: doi:10.1161/HYPERTENSIONAHA.106.084103
ISSN: 0194-911X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: There is convincing evidence that chronic treatment with N-acetyl-seryl-aspartyl-lysyl-proline (Ac-SDKP), a peptide normally found in tissues and biological fluids, reduces %%%collagen%%% deposition in

the heart and kidneys of hypertensive rats and rats with myocardial infarction. However, it is not known whether endogenous Ac-SDKP at basal concentrations has any physiological function related to %%%collagen%%% deposition. Prolyl oligopeptidase is responsible for release of Ac-SDKP from its precursor %%%thymosin%%%-beta(4). When we treated rats with a specific oral rolyl oligopeptidase inhibitor, Ac-SDKP decreased significantly in the plasma, heart, and kidney. In the present study, we tested the hypothesis that endogenous Ac-SDKP at basal levels plays a physiological role, antagonizing and/or preventing excessive %%%collagen%%% deposition. We studied whether chronic blockade of Ac-SDKP promotes %%%collagen%%% accumulation and/or accelerates this process in the presence of a profibrotic stimulus such as angiotensin II. We found that decreased basal levels of Ac-SDKP increased cardiac and renal perivascular fibrosis and promoted glomerulosclerosis. Moreover, in the presence of angiotensin II decreasing basal levels of Ac-SDKP accelerated interstitial cardiac fibrosis attributable to an increase in cells that produce %%%collagen%%%. We concluded that Ac-SDKP participates in the regulation of %%%collagen%%% content under normal conditions. We believe this is the first study showing that this peptide plays a physiological role at basal concentrations, preventing organ %%%collagen%%% accumulation.

0019606003 BIOSIS NO.: 200700265744
Decreased endogenous levels of Ac-SDKP increase organ fibrosis
AUTHOR: Cavasin Maria A (Reprint); Liao Tang-Dong; Yang Xiao-Ping; Carretero Oscar A
AUTHOR ADDRESS: Hypertens and Vasc Res Div, Henry Ford Hlth Syst, Detroit, MI USA**USA
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JOURNAL: Journal of Hypertension 24 (Suppl. 6): p305 DEC 2006 2006
CONFERENCE/MEETING: 21st Scientific Meeting of the International-Society-of-Hypertension/5th Asian-Pacific Congress of Hypertension/29th Annual Scientific Meeting of the Japanese-Society-of-Hypertension Fukuoka, JAPAN October 15 -19, 2006; 20061015
SPONSOR: Int Soc Hypertens
Japanese Soc Hypertens
ISSN: 0263-6352
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

1/7/11
DIALOG(R)File 5:Biosis Previews(R)

18999977 BIOSIS NO.: 200600345372
Effect of lactogenic stimuli and serotonin on secretory activation of bovine mammary epithelial cells (BMEC)
AUTHOR: Stiening Chad Michael (Reprint); Ben Abdallah Mheni; Hoying James B ; Collier Robert Joseph
AUTHOR ADDRESS: Univ Arizona, Tucson, AZ 85724 USA**USA
JOURNAL: FASEB Journal 20 (4, Part 1): pA616 MAR 6 2006 2006
CONFERENCE/MEETING: Experimental Biology 2006 Meeting San Francisco, CA, USA April 01 -05, 2006; 20060401
SPONSOR: Amer Assoc Anatomists
Amer Physiol Soc
Amer Soc Biochem & Mol Biol
Amer Soc Investigat Pathol
Amer Soc Nutr
Amer Soc Pharmacol & Expt Therapeut
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Culturing primary BMEC in a %%%collagen%%% gel with insulin-like growth factor I (IGF-I) and epidermal growth factor enables three-dimensional ductal growth and branching morphogenesis. We evaluated effects of structural (gel release) and hormonal (prolactin, PRL) stimuli on BMEC gene expression in presence of IGF-1 and cortisol. Gel release and PRL independently regulated a wide variety of genes involved in tissue remodeling, structural reorganization and lactogenesis. When the two stimuli were applied together, a smaller set of genes associated with secretory activation (caseins, alpha lactalburnin, calmodulin, xanthine dehydrogenase, galectin, milk lysozyme, galactokinase, Ig-family proteins, TGF-beta), intracellular trafficking (dynactin, spectrin, tubulin, dynein, %%%thymosin%%%-beta4, synaptophysin), epithelial tight junctions (claudin) and interactions (cadherins), and extracellular matrix remodeling (MMP, esterase) were regulated. We then evaluated effects of serotonin on BMEC secretion activation. We found that PRL upregulates TPH-1, the rate limiting enzyme for serotonin synthesis, and that serotonin down-regulates expression of cc-lactalbumin. Furthermore, when serotonin receptors are blocked with methysergide, transcription of both casein and a-lactalbumin is enhanced. A model is proposed whereby PRL induces the serotonin biosynthetic pathway, leading to negative

feedback of serotonin through its receptor(s) causing down-regulation of alpha-lactalbumin which reduces the rate of milk secretion.

1/7/12
DIALOG(R)File 5:Biosis Previews(R)

18911258 BIOSIS NO.: 200600256653
Differential gene expression in a paclitaxel-resistant clone of a head and neck cancer cell line
AUTHOR: Schmidt Marianne (Reprint); Schler Gabriele; Gruensfelder Petra; Hoppe Florian
AUTHOR ADDRESS: Univ Wurzburg, Dept Otorhinolaryngol, Josef Schneider Str 11, D-97080 Wurzburg, Germany**Germany
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JOURNAL: European Archives of Oto-Rhino-Laryngology 263 (2): p127-134 FEB 2006 2006
ISSN: 0937-4477
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The anti-neoplastic drug paclitaxel (taxol), which is known to block cells in the G2/M phase of the cell cycle through stabilization of microtubules, is meanwhile commonly used for chemotherapy of advanced head and neck cancer. Chemotherapy is primarily used in order to preserve laryngeal and/or pharyngeal structures. Although paclitaxel generally seems to be a powerful agent, it failed to reach a loco-regional tumor control in a sufficient percentage of patients. In order to investigate molecular resistance mechanisms, we have established a paclitaxel-resistant subline originating from the larynx carcinoma cell line HLaC79, which seemed to be partially dependent on taxol. The original and the descendant cell line were characterized by growth inhibition assays. We used western blotting and the cDNA subtraction (SSH) technique to identify genes differentially expressed in the taxol-resistant cell clone. cDNA subtraction revealed increased expression of six genes, including clathrin heavy chain, alpha 3-tubulin, a neuroblastoma-specific %%%Thymosin%%% beta, the ribosomal protein L7a, HLA-B associated transcript 3 and %%%collagen%%% III alpha 1 in the taxol-resistant cell line. Furthermore, western blots showed an overexpression of MDR-1 in the taxol-resistant clone, while alpha- and beta-tubulins and p48/IRF9 were expressed in equal amounts in both cell lines.

1/7/13
DIALOG(R)File 5:Biosis Previews(R)

18843714 BIOSIS NO.: 200600189109
Binding of PAI-1 to endothelial cells stimulated by %%%thymosin%%% beta 4 and modulation of their %%%fibrinolytic%%% potential
AUTHOR: Boncela Joanna; Smolarczyk Katarzyna; Wyroba Elzbieta; Cierniewski Czeslaw S (Reprint)
AUTHOR ADDRESS: Polish Acad Sci, Ctr Med Biol, PL-93232 Lodz, Poland** Poland
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JOURNAL: Journal of Biological Chemistry 281 (2): p1066-1072 JAN 13 2006 2006
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Our previous studies showed that %%%thymosin%%% beta 4 (T beta 4) induced the synthesis of plasminogen activator inhibitor-1 (PAI-1) in cultured human umbilical vein endothelial cells (HUVECs) via the AP-1 dependent mechanism and its enhanced secretion. In this work we provide evidence that the released PAI-1 is accumulated on the surface of HUVECs, exclusively in its active form, in a complex with alpha 1-acid glycoprotein (AGP) that is also up-regulated and released from the cells. This mechanism is supported by several lines of experiments, in which expression of both proteins was analyzed by flow cytometry and their colocalization supported by confocal microscopy. PAI-1 did not bind to quiescent cells but only to the T beta 4-activated endothelial cells. In contrast, significant amounts of AGP were found to be associated with the cells overexpressing enhanced green fluorescent protein (EGFP)-alpha 1-acid glycoprotein (AGP) without T beta 4 treatment. The AGP center dot PAI-1 complex was accumulated essentially at the basal surface of endothelial cells, and such cells showed (a) morphology characteristic for strongly adhered and spread cells and (b) significantly reduced plasmin formation. Taken together, these results provide the evidence supporting a novel mechanism by which active PAI-1 can be bound to the T beta 4-activated endothelial cells, thus influencing their %%%adhesive%%% properties as well as their ability to generate plasmin.

1/7/14
DIALOG(R)File 5:Biosis Previews(R)

18693896 BIOSIS NO.: 200600039291
Search for genes expressed during progression and recovery in the diseased kidney
AUTHOR: Monkawa Toshiaki (Reprint); Hayashi Matsuhiko
AUTHOR ADDRESS: Keio Univ, Sch Med, Dept Internal Med, Shinjuku Ku, Tokyo 1608582, Japan**Japan
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JOURNAL: Kidney International 68 (5): p1969-1970 NOV 2005 2005
CONFERENCE/MEETING: Meeting on Stem Cell and Regeneration of the Kidney Karuizawa, JAPAN January 20 -22, 2005; 20050120
SPONSOR: Int Soc Nephrol
ISSN: 0085-2538
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

1/7/15
DIALOG(R)File 5:Biosis Previews(R)

18611919 BIOSIS NO.: 200510306419
Glomerulosclerosis - Can proteomics help us understand it better? (vol 16, pg 2815, 2005)
AUTHOR: Anonymous
JOURNAL: Journal of the American Society of Nephrology 16 (11): p3446 NOV 2005 2005
ISSN: 1046-6673
DOCUMENT TYPE: Article; Errata
RECORD TYPE: Citation
LANGUAGE: English

1/7/16
DIALOG(R)File 5:Biosis Previews(R)

18584861 BIOSIS NO.: 200510279361
Autumn Meeting of the British-Society-for-Matrix-Biology, Bristol, ENGLAND, September 13 -14, 2004
AUTHOR: Anonymous
JOURNAL: International Journal of Experimental Pathology 86 (3): pA1-A56 JUN 2005 2005
CONFERENCE/MEETING: Autumn Meeting of the British-Society-for-Matrix-Biology Bristol, ENGLAND September 13 -14, 2004; 20040913

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Verigen
Promega
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Sigma Aldrich
Blackwell Med & Amaxa Biosyst
ISSN: 0959-9673
DOCUMENT TYPE: Meeting
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: This meeting of the British Society for Matrix Biology joint with the United Kingdom Tissue and Cell Engineering Society, University of Bristol, contains 79 abstracts in the English language. Clinical and non-clinical topics included stem cell therapy, tissue engineering, the skeletal system, osteoarthritis, vitiligo, metaphyseal chondrodysplasia, peripheral vascular disease, ischemia, Dupuytren's contraction, Sjorgen's syndrome and biochemistry.

1/7/17

18057499 BIOSIS NO.: 200400428288
Overexpression of the %%%thymosin%%% beta-4 gene is associated with increased invasion of SW480 colon carcinoma cells and the distant metastasis of human colorectal carcinoma
AUTHOR: Wang Wei-Shu; Chen Po-Min; Hsiao Hung-Liang; Wang Huann-Sheng; Liang Wen-Yih; Su Yeu (Reprint)
AUTHOR ADDRESS: Coll Life SciInst Biopharmaceut Sci, Natl Yang Ming Univ, Taipei, 112, Taiwan**Taiwan
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JOURNAL: Oncogene 23 (39): p6666-6671 August 26, 2004 2004
MEDIUM: print
ISSN: 0950-9232 _(ISSN print)
DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Cell-matrix and cell-cell %%%adhesive%%% interactions play important roles in the normal organization and stabilization of the cell layer in epithelial tissue. Alterations in the expression and function of these adhesion systems that cause a switch to a migratory phenotype in tumor invasion and metastasis are critical for the malignant conversion of epithelial cells. %%%Thymosin%%% beta-4 (Tbeta-4) is the major actin-sequestering protein that has been shown to be upregulated in a wide variety of human carcinomas and has been implicated to be involved in altering the motility of certain tumors. We have recently demonstrated that the growth rate, colony formation in soft agar, and motility, all good indicators for malignant progression, of SW480 colon carcinoma cells are dramatically increased by enforced Tbeta-4 expression. To test the hypothesis that overexpression of this G- actin sequestering peptide also promotes tumor invasion, we examined not only the invasion capability of Tbeta-4-overexpressing SW480 cells, but also the expression levels of Tbeta-4 as well as several proteins that participate in different stages of tumor progression in matched samples of human primary colorectal adenocarcinoma and liver metastases from several patients. A marked increase on the invasiveness in Tbeta-4-overexpressing SW480 cells with increased levels and activity of matrix metalloproteinase-7 (MMP-7) was observed. Furthermore, the levels of Fas as well as the susceptibility to Fas ligand-mediated apoptosis in Tbeta-4-overexpressing cells were significantly decreased. Interestingly, the levels of Tbeta-4 mRNA, beta-catenin, c-Myc, and MMP-7 in metastatic liver lesions were relatively higher, whereas the levels of E-cadherin and Fas were significantly lower than those in the matched primary colorectal tumors. These results suggest that upregulation of Tbeta-4, by promoting the disruption of cell-cell adhesion and a consequential activation of the beta-catenin signaling, could be a key event in the acquisition of growth advantages as well as invasive phenotypes in human colorectal carcinomas.

1/7/18
DIALOG(R)File 5:Biosis Previews(R)

18046580 BIOSIS NO.: 200400417369
Factor XIIIa incorporates %%%thymosin%%% beta4 preferentially into the %%%fibrin%%%(ogen) alphaC-domains
AUTHOR: Makogonenko Evgeny; Goldstein Allan L; Bishop Paul D; Medved Leonid (Reprint)
AUTHOR ADDRESS: Dept BiochemJerome H Holland Lab Biomed Sci, Amer Red Cross, Rockville, MD, 20855, USA**USA

AUTHOR E-MAIL ADDRESS: medvedL@usa.redcross.org
JOURNAL: Biochemistry 43 (33): p10748-10756 August 24, 2004 2004
MEDIUM: print
ISSN: 0006-2960 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: It was shown recently that tissue transglutaminase and presumably plasma transglutaminase, factor XIIIa, can covalently incorporate into %%%fibrin%%%(ogen) a physiologically active peptide, %%%thymosin%%% beta4 ((Huff et al. (2002) FASEB J. 16, 691-696). To clarify the mechanism of this incorporation, we studied the interaction of %%%thymosin%%% beta4 with %%%fibrinogen%%%, %%%fibrin%%%, and their recombinant fragments, the gamma-module (gamma-chain residues 148-411), and the alphaC-domain (Aalpha-chain residues 221-610) and its truncated variants by immunoblot and ELISA. No significant noncovalent interaction between them was detected in the absence of activated factor XIII, while in its presence %%%thymosin%%% beta4 was effectively incorporated into %%%fibrin%%% and to a lesser extent into %%%fibrinogen%%%. The incorporation at physiological concentrations of %%%fibrin%%%(ogen) and factor XIII was significant with molar incorporation ratios of %%%thymosin%%% beta4 to %%%fibrinogen%%% and %%%fibrin%%% of 0.2 and 0.4, respectively. Further experiments revealed that although activated factor XIII incorporates %%%thymosin%%% beta4 into the isolated gamma-module and alphaC-domain, in %%%fibrin%%% the latter serves as the major incorporation site. This site was further localized to the COOH-terminal portion of the alphaC-domain including residues 392-610.

1/7/19

17737562 BIOSIS NO.: 200400107268
The profile of gene expression of human marrow mesenchymal stem cells.
AUTHOR: Silva Wilson A; Covas Dimas T; Panepucci Rodrigo A; Proto-Siqueira Rodrigo; Siufi Jorge L C; Zanette Dalila L; Santos Anemari R D; Zago Marco A (Reprint)
AUTHOR ADDRESS: Hemocentro, R. Tenente Catao, Roxo, 2501, 14051-140, Ribeirao Preto, Brazil**Brazil
AUTHOR E-MAIL ADDRESS: marazago@usp.br
JOURNAL: Stem Cells (Miamisburg) 21 (6): p661-669 2003 2003
MEDIUM: print
ISSN: 1066-5099 _(ISSN print)
DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Mesenchymal stem cells (MSCs) are multipotent precursors present in adult bone marrow, that differentiate into osteoblasts, adipocytes and myoblasts, and play important roles in hematopoiesis. We examined gene expression of these cells by serial analysis of gene expression, and found that %%%collagen%%% I, secreted protein acidic and rich in cysteine (osteonectin), transforming growth factor beta- (TGF-beta) induced, cofilin, galectin-1, laminin-receptor 1, cyclophilin A, and matrix metalloproteinase-2 are among the most abundantly expressed genes. Comparison with a library of CD34+ cells revealed that MSCs had a larger number of expressed genes in the categories of cell adhesion molecule, extracellular and development. The two types of cells share abundant transcripts of many genes, some of which are highly expressed in myeloid progenitors (%%%thymosin%%%-beta4 and beta10, fos and jun). Interleukin-11 (IL-11), IL-15, IL-27 and IL-10R, IL-13R and IL-17R were the most expressed genes among the cytokines and their receptors in MSCs, and various interactions can be predicted with the CD34+ cells. MSCs express several transcripts for various growth factors and genes suggested to be enriched in stem cells. This study reports the profile of gene expression in MSCs and identifies the important contribution of extracellular protein products, adhesion molecules, cell motility, TGF-beta signaling, growth factor receptors, DNA repair, protein folding, and ubiquination as part of their transcriptome.

17692231 BIOSIS NO.: 200400062988
Genomic and proteomic mechanisms of increased vascular stiffness in aging monkey aorta.
AUTHOR: Depre Christophe (Reprint); Yan Lin (Reprint); Yang Guiping (Reprint); Markiewicz Jadwiga (Reprint); Peppas Athanasios (Reprint); Kim Song-Jung (Reprint); Vatner Stephen F (Reprint)
AUTHOR ADDRESS: UMDNJ, Newark, NJ, USA**USA
JOURNAL: Circulation 108 (17 Supplement): pIV-68-IV-69 October 28, 2003 2003
MEDIUM: print
CONFERENCE/MEETING: American Heart Association Scientific Sessions 2003 Orlando, FL, USA November 09-12, 2003; 20031109
SPONSOR: American Heart Association
ISSN: 0009-7322 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract

RECORD TYPE: Citation
LANGUAGE: English

1/7/21
DIALOG(R)File 5:Biosis Previews(R)

17442818 BIOSIS NO.: 200300401537
Genomic changes with increased vascular stiffness in aging monkey aorta.
AUTHOR: Depre Christophe (Reprint); Markiewicz Jadwiga; Yan Lin; Yang Guiping; Pappas Atanassios; Shen You-tang; Kim Song-Jung; Vatner Stephen F
AUTHOR ADDRESS: Cell Biology, University of Medicine New Jersey, 185 south orange avenue, G-609, Newark, NJ, 07103, USA**USA
AUTHOR E-MAIL ADDRESS: deprech@umdnj.edu; markiewicz@umdnj.edu; yanli@umdnj.edu; yanggu@umdnj.edu; pappasat@umdnj.edu; shen@merck.org; kimsong@umdnj.edu; vatnersfo@umdnj.edu
JOURNAL: FASEB Journal 17 (4-5): pAbstract No. 258.8 March 2003 2003
MEDIUM: e-file
CONFERENCE/MEETING: FASEB Meeting on Experimental Biology: Translating the Genome San Diego, CA, USA April 11-15, 2003; 20030411
SPONSOR: FASEB
ISSN: 0892-6638 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Increased vascular stiffness in aging humans results in increased cardiac workload and dysfunction. To better determine the molecular mechanisms involved in this process, we compared by subtractive hybridization the genomic profile of the aorta in young (4 yrs) vs old (20 yrs) monkeys (M. Macaca, n=3 per group). Medial thickness increased from 200+-10 to 315+-10 im in young vs old monkeys, respectively (P<0.05), which results in increased stiffness, calculated from direct and continuous measurement of aortic pressure and diameter. There was no evidence of atherosclerosis. mRNA was purified from transmural samples harvested in the thoracic aorta, reverse-transcribed in double-stranded DNA and submitted to subtractive hybridization. The identity of the regulated genes was determined by sequencing. Aorta from old monkeys showed an upregulation of genes participating in vascular smooth muscle cell motility (alpha-actin, %%%thymosin%%%-4 beta, vimentin), as well as an upregulation of genes involved in protein trafficking and secretion (Raf, Rab, Ran binding-protein, Rac 1). Expression of %%%collagen%%% type 1 was also upregulated, which correlated with a 2.2-fold increase in aortic %%%collagen%%% content in old vs young samples (P<0.05).

Therefore, increased stiffness with aging in the non-human primate correlates at the molecular level with a genomic pattern consistent with a migratory and secretory pattern of vascular smooth muscle cells, as well as an accumulation of %%%collagen%%%.

1/7/22
DIALOG(R)File 5:Biosis Previews(R)

17337564 BIOSIS NO.: 200300295383
Gene array analysis of differential gene expression in human hearts with dilated cardiomyopathy.
AUTHOR: Zimmermann Rene (Reprint); Donay Andrea; Kostin Sawa; Hein Stefan; Boengler Kerstin; Kloevekorn Wolf-Peter; Schaper Jutta
AUTHOR ADDRESS: Vascular Genomics, Kerckhoff-Clinic, Benekestr. 2-8, Bad Nauheim, 61231, Germany**Germany
AUTHOR E-MAIL ADDRESS: r.zimmer@vascular-genomics.de; a.donay@kerckhoff.mpg.de; s.kostin@kerckhoff.mpg.de; s.hein@kerckhoff.mpg.de; k.boengler@kerckhoff.mpg.de; w-p.kloevekorn@kerckhoff.med.uni-giessen.de; j.schaper@kerckhoff.mpg.de
JOURNAL: FASEB Journal 17 (4-5): pAbstract No. 345.18 March 2003 2003
MEDIUM: e-file
CONFERENCE/MEETING: FASEB Meeting on Experimental Biology: Translating the Genome San Diego, CA, USA April 11-15, 2003; 20030411
SPONSOR: FASEB
ISSN: 0892-6638 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English
ABSTRACT: We performed gene array analysis of mRNA from 6 DCM hearts and 4 healthy ones to elucidate the molecular basis of DCM. Differential expression was confirmed by Northern (N) and Western (W) blot analysis. Besides upregulation of genes known to be involved in DCM (ANP, fibronectin, %%%collagen%%% I, annexin VI) we found an upregulation of %%%thymosin%%% ss-4 and cofilin (2-fold; N,W), both involved in actin depolymerization. Myocardial remodeling was shown by the downregulation of plakoglobin (2-fold; W) and regulatory MLC. Altered susceptibility to apoptosis is reflected in the downregulation of anti-apoptotic genes (TERT and NF-ATC) and in the upregulation of ID3 (1.7-fold; W). The expression of genes with a possible protective function was altered, i.e. upregulation of integrin-ss (2-fold; W), annexin I (4-fold; W), downregulation of phospholipase A2 receptor, RANTES, LFA-1 alpha, Kallistatin. Carp, a nuclear protein controling the regulation of other muscle specific genes, is significantly upregulated (N;W). Increased protein turnover in DCM is indicated by the fact that N-cadherin was

upregulated 2.3-fold in N, remained unchanged in W and revealed a difference in the distribution pattern. Our results point to an imbalance of damaging and protective mechanisms in DCM. Gene arrays may contribute significantly to the understanding of molecular pathways underlying this imbalance.

1/7/23
DIALOG(R)File 5:Biosis Previews(R)

17223269 BIOSIS NO.: 200300181988
%%%Thymosin%%% beta4 and a synthetic peptide containing its actin-binding domain promote dermal wound repair in db/db diabetic mice and in aged mice.
AUTHOR: philp Deborah; Badamchian Mahnaz; Scheremeta Brooke; Nguyen Mychi; Goldstein Allan L; Kleinman Hynda K (Reprint)
AUTHOR ADDRESS: NIH, NIDCR, 30 Convent Dr, 30/433, MSC 4370, Bethesda, MD, 20892-4370, USA**USA
AUTHOR E-MAIL ADDRESS: hkleinman@dir.nidcr.nih.gov
JOURNAL: Wound Repair and Regeneration 11 (1): p19-24 January-February 2003 2003
MEDIUM: print
ISSN: 1067-1927
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Impaired wound healing is a problem for immobilized patients, diabetics, and the elderly. %%%Thymosin%%% beta4 has previously been found to promote dermal and corneal repair in normal rats. Here we report that %%%thymosin%%% beta4 was also active in accelerating wound repair in full-thickness dermal wounds in both db/db diabetic and aged mice. We found that %%%thymosin%%% beta4 in either phosphate-buffered saline or a hydrogel formulation is active in promoting dermal wound repair in normal rats. In diabetic mice, where healing is delayed, we found that wound contracture and %%%collagen%%% deposition were significantly increased in the mice treated with %%%thymosin%%% beta4 in either phosphate buffered saline solution or a hydrogel formulation. No difference was observed in keratinocyte migration, with all of the diabetic animals showing almost complete coverage of the wound at 8 days. Wound healing in 26-month-old (aged) animals was significantly delayed. %%%Thymosin%%% beta4 accelerated wound healing in these aged mice, with increases in keratinocyte migration, wound contracture, and %%%collagen%%% deposition. The hydrogel formulation generally showed similar wound healing activity with %%%thymosin%%% beta4 in PBS. The actin-binding domain of

%%%thymosin%%% beta4 duplicated in a seven-amino acid synthetic peptide, LKKTETQ, was able to promote repair in the aged animals comparable to that observed with the parent molecule. These studies show that %%%thymosin%%% beta4 is active for wound repair in models of impaired healing and may have efficacy in chronic wounds in humans.

1/7/24
DIALOG(R)File 5:Biosis Previews(R)

17078350 BIOSIS NO.: 200300037069
Antimicrobial peptides from human platelets.
AUTHOR: Tang Yi-Quan; Yeaman Michael R; Selsted Michael E (Reprint)
AUTHOR ADDRESS: Department of Pathology, College of Medicine, University of California, Irvine, CA, 92697-4800, USA**USA
AUTHOR E-MAIL ADDRESS: meselste@uci.edu
JOURNAL: Infection and Immunity 70 (12): p6524-6533 December 2002 2002
MEDIUM: print
ISSN: 0019-9567 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Platelets share structural and functional similarities with granulocytes known to participate in antimicrobial host defense. To evaluate the potential antimicrobial activities of platelet proteins, normal human platelets were stimulated with human thrombin in vitro. Components of the stimulated-platelet supernatants were purified to homogeneity by reversed-phase high-performance liquid chromatography. Purified peptides with inhibitory activity against Escherichia coli ML35 in an agar diffusion antimicrobial assay were characterized by mass spectrometry, amino acid analysis, and sequence determination. These analyses enabled the identification of seven thrombin-releasable antimicrobial peptides from human platelets: platelet factor 4 (PF-4), RANTES, connective tissue activating peptide 3 (CTAP-3), platelet basic protein, %%%thymosin%%% beta-4 (Tbeta-4), %%%fibrinopeptide%%% B (FP-B), and %%%fibrinopeptide%%% A (FP-A). With the exception of FP-A and FP-B, all peptides were also purified from acid extracts of nonstimulated platelets. The in vitro antimicrobial activities of the seven released peptides were further tested against bacteria (E. coli and Staphylococcus aureus) and fungi (Candida albicans and Cryptococcus neoformans). Each peptide exerted activity against at least two organisms. Generally, the peptides were more potent against bacteria than fungi, activity was greater at acidic pHs, and antimicrobial activities were dose dependent. Exceptions to these observations were observed with PF-4, which displayed

a bimodal dose-response relationship in microbicidal assays, and Tbeta-4, which had greater activity at alkaline pHs. At concentrations at which they were individually sublethal, PF-4 and CTAP-3 exerted synergistic microbicidal activity against E. coli. Collectively, these findings suggest a direct antimicrobial role for platelets as they are activated to release peptides in response to trauma or mediators of inflammation.

1/7/25
DIALOG(R)File 5:Biosis Previews(R)

16716648 BIOSIS NO.: 200200310159
%%%Thymosin%%% beta4 is released from human blood platelets and attached by factor XIIIa (transglutaminase) to %%%fibrin%%% and %%%collagen%%%
AUTHOR: Huff Thomas (Reprint); Otto Angela M; Mueller Christian S G; Meier Markus; Hannappel Ewald
AUTHOR ADDRESS: Faculty of Medicine, Institute of Biochemistry, University Erlangen-Nuremberg, Fahrstr. 17, 91054, Erlangen, Germany**Germany
JOURNAL: FASEB Journal 16 (7): p691-696 May, 2002 2002
MEDIUM: print
ISSN: 0892-6638
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The beta-thymosins constitute a family of highly conserved and extremely water-soluble 5 kDa polypeptides. %%%Thymosin%%% beta4 is the most abundant member; it is expressed in most cell types and is regarded as the main intracellular G-actin sequestering peptide. There is increasing evidence for extracellular functions of %%%thymosin%%% beta4. For example, %%%thymosin%%% beta4 increases the rate of attachment and spreading of endothelial cells on matrix components and stimulates the migration of human umbilical vein endothelial cells. Here we show that %%%thymosin%%% beta4 can be cross-linked to proteins such as %%%fibrin%%% and %%%collagen%%% by tissue transglutaminase. %%%Thymosin%%% beta4 is not cross-linked to many other proteins and its cross-linking to %%%fibrin%%% is competed by another family member, %%%thymosin%%% beta10. After activation of human platelets with thrombin, %%%thymosin%%% beta4 is released and cross-linked to %%%fibrin%%% in a time- and calcium-dependent manner. We suggest that %%%thymosin%%% beta4 cross-linking is mediated by factor XIIIa, a transglutaminase that is coreleased from stimulated platelets. This provides a mechanism to increase the local concentration of %%%thymosin%%% beta4 near sites of clots and tissue damage, where it may contribute to wound healing, angiogenesis and inflammatory responses.

1/7/26
DIALOG(R)File 5:Biosis Previews(R)

16073640 BIOSIS NO.: 200100245479
Characterization and phylogenetic analysis of Brachytury-downstream genes in Ciona intestinalis embryo
AUTHOR: Hotta Kohji (Reprint); Takahashi Hiroki; Satou Yutaka (Reprint); Gojobori Takashi; Satoh Noriyuki (Reprint)
AUTHOR ADDRESS: Dept. of Zool., Grad. Sch. of Sci., Kyoto Univ., Kyoto, Japan**Japan
JOURNAL: Genes and Genetic Systems 75 (6): p364 December, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 72nd Annual Meeting of the Genetics Society of Japan Kyoto, Japan November 03-05, 2000; 20001103
SPONSOR: Genetics Society of Japan
ISSN: 1341-7568
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

1/7/27
DIALOG(R)File 5:Biosis Previews(R)

15785544 BIOSIS NO.: 200000503857
Functional analysis of human %%%thymosin%%% beta4 promoter in aortic valve interstitial cells
AUTHOR: Li Q Y (Reprint); Lafferty R P (Reprint); Levy R J (Reprint)
AUTHOR ADDRESS: Cardiology Res, Children's Hosp Philadelphia, Abramson Bldg, Philadelphia, PA, USA**USA
JOURNAL: American Journal of Human Genetics 67 (4 Supplement 2): p180 October, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 50th Annual Meeting of the American Society of Human Genetics Philadelphia, Pennsylvania, USA October 03-07, 2000; 20001003
SPONSOR: American Society of Human Genetics
ISSN: 0002-9297
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

1/7/28
DIALOG(R)File 5:Biosis Previews(R)

15658518 BIOSIS NO.: 200000376831
The EST analysis of kidney cells from Japanese flounder Paralichthys olivaceus injected with beta-glucan
AUTHOR: Kono Tomoya (Reprint); Sakai Masahiro (Reprint)
AUTHOR ADDRESS: Faculty of Agriculture, Miyazaki University, Miyazaki, 889-21, Japan**Japan
JOURNAL: Developmental and Comparative Immunology 24 (Supplement 1): pS72 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 8th Congress of the International Society of Developmental and Comparative Immunology Cairns, Australia July 03-06, 2000; 20000703
ISSN: 0145-305X
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

1/7/29
DIALOG(R)File 5:Biosis Previews(R)

15225147 BIOSIS NO.: 199900484807
%%%Thymosin%%% beta4 accelerates wound healing
AUTHOR: Malinda Katherine M; Sidhu Gurmel S; Mani Haresh; Banaudha Krishna; Maheshwari Radha K; Goldstein Allan L; Kleinman Hynda K (Reprint)
AUTHOR ADDRESS: Laboratory of Developmental Biology, Institute of Dental and Craniofacial Research, National Institutes of Health, 30 Convent Drive, MSC 4370, Building 30, Room 407, Bethesda, MD, 20892-4370, USA** USA
JOURNAL: Journal of Investigative Dermatology 113 (3): p364-368 Sept., 1999 1999
MEDIUM: print
ISSN: 0022-202X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Angiogenesis is an essential step in the repair process that occurs after injury. In this study, we investigated whether the angiogenic thymic peptide %%%thymosin%%% beta4 (Tbeta4) enhanced wound healing in a rat full thickness wound model. Addition of Tbeta4 topically

or intraperitoneally increased reepithelialization by 42% over saline controls at 4 d and by as much as 61% at 7 d post-wounding. Treated wounds also contracted at least 11% more than controls by day 7. Increased %%%collagen%%% deposition and angiogenesis were observed in the treated wounds. We also found that Tbeta4 stimulated keratinocyte migration in the Boyden chamber assay. After 4-5 h, migration was stimulated 2-3-fold over migration with medium alone when as little as 10 pg of Tbeta4 was added to the assay. These results suggest that Tbeta4 is a potent wound healing factor with multiple activities that may be useful in the clinic.

1/7/30
DIALOG(R)File 5:Biosis Previews(R)

13561329 BIOSIS NO.: 199699195389
Modulation of in vitro and in vivo hemostatic reactions by representatives of regulatory peptide families
AUTHOR: Ashmarin I P; Lyapina L A; Pastorova V E
AUTHOR ADDRESS: Dep. Hum. Anim. Physiol., Fac. Biol., M.V. Lomonosov Mosc. State Univ., Moscow, Russia**Russia
JOURNAL: Vestnik Rossiiskoi Akademii Meditsinskikh Nauk 0 (6): p50-57 1996
1996
ISSN: 0869-6047
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: Russian

ABSTRACT: Data available in the literature and the author's own findings of the effects of regulatory peptide (RP) and their analogues are summarized. MIF, TRH, and its analog PR-546, the paraopioid RP, leuenkephalin, dalargin, the ACTH analogue Semax, tafcin, thymosine, interleukin- 1, vasopressin, oxytocin, bradykinin, defencin, and some proline-containing oligopeptides, such as Pro-Gly, Gly-Pro, Trp-Pro, Pro-Gly-Pro, Gly-Pro-Gly-Gly were studied. A complex of in vitro and in vivo tests identified three groups of RP: 1) neutral ones as to the hemostatic reactions studied; 2) stimulants of hypercoagulation and %%%fibrin%%% polymerization; 3) inhibitors of blood coagulation, increased %%%fibrinolysis%%%, and %%%fibrin%%% demopolymerization. The %%%fibrinolytic%%% and antithrombotic effects of Semax (in vivo), the procoagulative action of defencin, and the enhanced anticoagulant effects in the combinations of Semax-heparin and tafcin (in vivo) attract particular attention. Semax alone and in combination with heparin is recommended for clinical studies in respective hemostatic abnormalities.

1/7/31
DIALOG(R)File 5:Biosis Previews(R)

12063632 BIOSIS NO.: 199497084917
The platelet cytoskeleton
AUTHOR: Fox Joan E B
AUTHOR ADDRESS: Children's Hosp. Oakland Res. Inst., 747 52nd Street, Oakland, CA 94609, USA**USA
JOURNAL: Thrombosis and Haemostasis 70 (6): p884-893 1993 1993
ISSN: 0340-6245
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The platelet cytoskeleton contains two actin filament-based components. One is the cytoplasmic actin filaments which fill the cytoplasm and mediate contractile events. The other is the membrane skeleton, which coats the plasma membrane and regulates properties of the membrane such as its contours and stability. In the unstimulated platelet, only 30-40% of the actin is polymerized into filaments; the rest is thought to be prevented from polymerizing by the association of %%%thymosin%%% 04 with monomeric actin and by the association of gelsolin with the barbed ends of pre-existing actin filaments. When platelets are activated, there is a rapid increase in actin polymerization; new filaments fill the extending filopodia and form a network at the periphery of the platelet. As a result of activation, myosin binds to cytoplasmic actin filaments, causing them to move towards the center of the platelet. As platelets aggregate, additional cytoskeletal reorganizations occur: GP IIb-IIIa associates with %%%adhesive%%% ligand in a platelet aggregate; this results in the association of GP IIb-IIIa, membrane skeleton proteins, and signaling molecules with cytoplasmic actin. Future studies should help to elucidate the significance of the cytoskeleton in regulating signal transduction events in platelets.

1/7/32
DIALOG(R)File 5:Biosis Previews(R)

10625165 BIOSIS NO.: 199191008056
EFFECT OF %%%THYMOSIN%%% PEPTIDES ON THE SYSTEM OF HEMOSTASIS
AUTHOR: LYAPINA L A (Reprint); PASTOROVA V E; KUDRYASHOV B A; ZAZHIREI V D; SOKOLOVA E A; LIVSHITS V A; ASHMARIN I P
AUTHOR ADDRESS: BIOL FAC, MV LOMONOSOV MOSC STATE UNIV, MOSCOW, USSR**USSR

JOURNAL: Izvestiya Akademii Nauk SSSR Seriya Biologicheskaya (3): p377-382
1990
ISSN: 0002-3329
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: RUSSIAN

ABSTRACT: Repeated introduction of thymus polypeptide drug thymoptin (8 times every 24 hours, 1 mg/kg) led to the increase in total and non-enzymatic %%%fibrinolytic%%% activity of blood plasma and the decrease in %%%fibrinogen%%% concentration. Thymoptin could lyse non-stabilized %%%fibrin%%% and cause depolymerization of aggregates of %%%fibrin%%% monomer in vitro. Highest depolymerization activity was observed at thymoptin concentration of 10 mg/ml.

1/7/33
DIALOG(R)File 5:Biosis Previews(R)

09998841 BIOSIS NO.: 199039052230
THE BACTERIAL TOXIN FROM PASTEURELLA MULTOCIDA INDUCES CHANGES IN THE PHENOTYPE OF THE ROS 17-2.8 RAT OSTEOSARCOMA CELL
AUTHOR: STERNER A (Reprint); LANSKE B; KAESLER S; HESCH R-D; ATKINSON M J; UEBERSCHAER S
AUTHOR ADDRESS: ABT PATHOL, TIERAERTZLICHE HOCHSCHULE HANNOVER, HANNOVER, W GER**WEST GERMANY
JOURNAL: Acta Endocrinologica Supplementum 122 (1): p14 1990
CONFERENCE/MEETING: THIRTY-FOURTH SYMPOSIUM OF THE GERMAN SOCIETY OF ENDOCRINOLOGY, HANNOVER, WEST GERMANY, MARCH 14-17, 1990. ACTA ENDOCRINOL SUPPL.
ISSN: 0300-9750
DOCUMENT TYPE: Meeting
RECORD TYPE: Citation
LANGUAGE: ENGLISH

1/7/34
DIALOG(R)File 5:Biosis Previews(R)

08857526 BIOSIS NO.: 198834086417
THYMIC HORMONES INHIBIT WOUND HEALING
AUTHOR: BARBUL A (Reprint); KNUD-HANSEN J; WASSERKRUG H L; EFRON G
AUTHOR ADDRESS: DEP SURGERY, SINAI HOSP, BALTIMORE, MD 21215, USA**USA

JOURNAL: Surgical Forum (Chicago) 38 p34-36 1987
CONFERENCE/MEETING: 43RD ANNUAL SESSIONS OF THE FORUM ON FUNDAMENTAL SURGICAL PROBLEMS HELD AT THE 73RD ANNUAL CLINICAL CONGRESS, SAN FRANCISCO, CALIFORNIA, USA, OCTOBER 1987. SURG FORUM.
ISSN: 0071-8041
DOCUMENT TYPE: Meeting
RECORD TYPE: Citation
LANGUAGE: ENGLISH
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$1.86 TELNET
$90.19 Estimated cost this search
$90.19 Estimated total session cost 1.249 DialUnits
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